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AUTHOR(S):

Saito, Masami

CITATION:

Saito, Masami. Auditory Disturbances in Course of Aphasia. 音声科学研究 1964, 3: 66-75

ISSUE DATE:

1964

URL:

<http://hdl.handle.net/2433/52624>

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Auditory Disturbances in Course of Aphasia

Masami SAITO

Before passing to the account of present observations, the author may give a brief history of thought about aphasia.

Since the earliest times, speech disorders had been so familiar to physicians that the word "aphasia" was first employed by Sextus Empiricus about eighteen hundreds years ago, when he used it not in a pathological sense but in a philosophical sense.

Almost all the clinical features of aphasia have been described before 1800 by many enlightened persons, though it seems that they have neither drawn any conclusion from their experiences. In 1861, Broca demonstrated the brains of two patients with loss of speech, concluding that aphemia, the loss of speech, was the results of an accurately circumscribed lesion of the posterior third of the second and third frontal convolutions. Three years later, Trousseau employed the term "aphasia" to replace aphemia. Since then the term aphasia has been generally used to indicate the disturbances of speech. When aphasia strictly interpreted means absence of speech, the term dysphasia is frequently employed.

Wernicke in 1874 located the center for auditory images in the left first temporal convolution and distinguished three varieties of aphasia; sensory, due to destruction of this center, motor, due to a lesion of Broca's area, and a third due to interference with conduction between these two centers.

The main line of development of thought about aphasia in nineteenth century was in the direction of increasing localization of function. Many investigators have made important contributions to this field and set out their own classifications of aphasia preparing diagrams to illustrate the anatomical basis of speech, reading and writing.

Exponents of a dynamic view point, however, were not lacking. Jackson was interested mainly in the dynamic aspects of speech and pointed out that words were required for thinking, for most of our thinking at least, but the speechless man was not wordless; there was an automatic and unconscious or subconscious service of words. Freud, much influenced by Jackson, strongly criticized the "diagram makers" pointing out that their classifications corresponded to neither the clinical nor the pathological facts. Head maintained that the localizationists failed to appreciate that the logical formulae of the intellect do not correspond absolutely to physical events and that the universe does not exist as an exercise for the human mind.

It is impossible and unnecessary here to introduce fully all the views on aphasia. The contribution of Marie, however, must not be neglected, because he is the first person attacked the long accepted conception of Wernicke in his paper titled "The third frontal convolution does not play any role in the function of language". He contended that there was only one form of aphasia, sensory aphasia, which was not a special loss of word images but a defect of general intelligence and of special intelligence of language.

Bay claims that various forms of aphasias are a heterogenous collection of speech disorders with very different pathology. By recording tongue movements, he has revealed that the patients with motor aphasia exhibit disturbance of the muscular movement in speaking which leads to a dysarthria with a characteristic phonetic desintegration. He denies the essential difference between sensory and amnesic aphasia in the sphere of speech function. Therefore he maintains that the speech disorder is identical.

By recording pure tone audiograms for patients suffering from aphasias, Alajouanine and his collaborators have suggested that the results confirm the Marie's contention.

The present author has also studied auditory disturbances in course of aphasia on twenty-three selected cases of speech disorders with the aid of clinical audiometer and obtained the following results which are not always consistent with those of Alajouanine et al. In this paper, the properties of auditory disorders and their significance in aphasia have been reported and discussed.

MATERIALS AND METHODS

Of many patients with speech disorders who had come under the author's care in neuropsychiatric wards of the Kyoto University Medical School Hospital and of the Kansai Medical School Hospital since 1955, twenty-three cases were selected for this study. Except for three patients in their fifties, all the remaining twenty cases are under 50 years of age for the purpose of excluding influences from presbycusis. In the study group, the age distribution ranges from 17 to 58 years with an average of 38. They have no history of family deafness, antenatal and postnatal disease and no significant abnormality on examination of the ears, nose and throat.

Twelve of them are clinically classified as motor type, eight as sensory and three as amnesic types at the beginning of this study.

Preliminary assessment of auditory threshold was carried out for continuous pure tone in the sound treated room at the Department of Otorhinolaryngology, University of Kyoto, and at the otorhinolaryngological wards of the Kansai Medical School Hospital.

RESULTS

Successive composite audiograms with continuous pure tone for the subjects of motor aphasia have shown more or less initial deteriorations over practically the whole of the tested frequency range, though there is a marked individual variation. From the types of threshold rise the author distinguishes three varieties:

1) Low tone loss; seven cases showed a threshold rise much more prominent in low and middle frequencies than in high frequency. An average loss of low and middle tone is in the region of 40 db., from 2, to 4,000 c.p.s. of order of 20 db. and above 4,000 from 5 to 15 db.

2) High tone loss; one sustained a steeper initial loss for frequencies above 2,000 c.p.s. than for low frequency range.

3) Nearly normal hearing; four patients presented almost flat audiograms with the threshold rise of 10 db. or less.

The average loss for speech frequencies in group 1 in region of 40 db. while in group 2 and 3 less than 10 db.

The patients with sensory aphasia have shown audiograms resembling those of bilateral perceptive deafness which is characterized by the threshold rise for high frequency range. There is also a considerable variation in individual audiograms but a tendency towards an extremely severe loss for frequencies above 2,000 c.p.s. is in common to all. It is well illustrated in Figure 1, a bone conducted audiogram for continuous pure tone obtained in a patient showing a typical pure word-deafness lasting just over ten years.

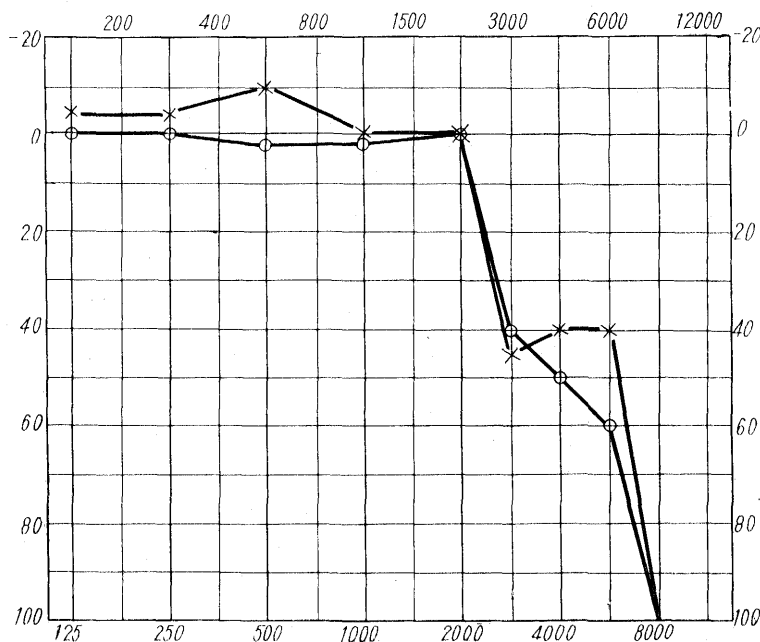


Fig. 1

The whole of three cases of amnesic aphasia has presented normal hearing for the routine assessment either by air conduction or bone conduction.

Some of the patients presented noticeable threshold rise on the routine assessment were issued a hearing aid and followed up, but their audiograms remained almost unchanged.

It must be noted, however, that further examination with interrupted pure tone in place of continuous pure tone have revealed an interesting property of the threshold rise found in sensory and motor aphasias. The hearing loss for continuous tone is often improved or sometimes disappeared and the audiograms by interruption test tends to be flatter. Figure 2 shows the audiograms of a

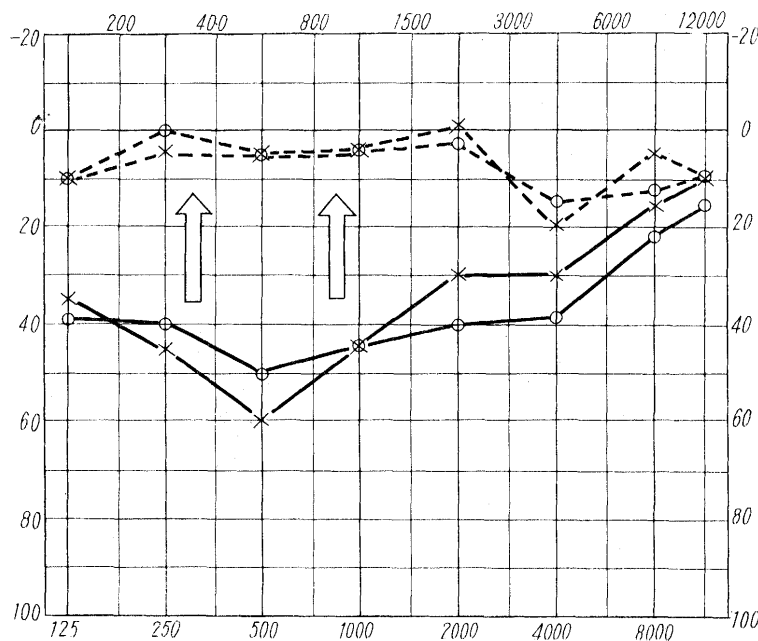


Fig. 2

patient suffering from a cortical motor aphasia of vascular origin; full lines show the results of continuous pure tone test and broken lines show those of interrupted pure tone test.

Loss of hearing was repeatedly assessed in four cases in order to estimate the temporary threshold shift. All of them have presented more or less different results whenever the assessment is repeated in the time interval of different orders. It may be said that the threshold rise in these patients is very instable and not always fixed. Figure 3 shows well this oscillation of auditory function; shaded area illustrates the extent of threshold oscillations.

During the observations, four patients have presented the modification of clinical forms of their speech disorders. One of the patients had been classified as sensory aphasia showed a marked improvement in comprehension and misap-

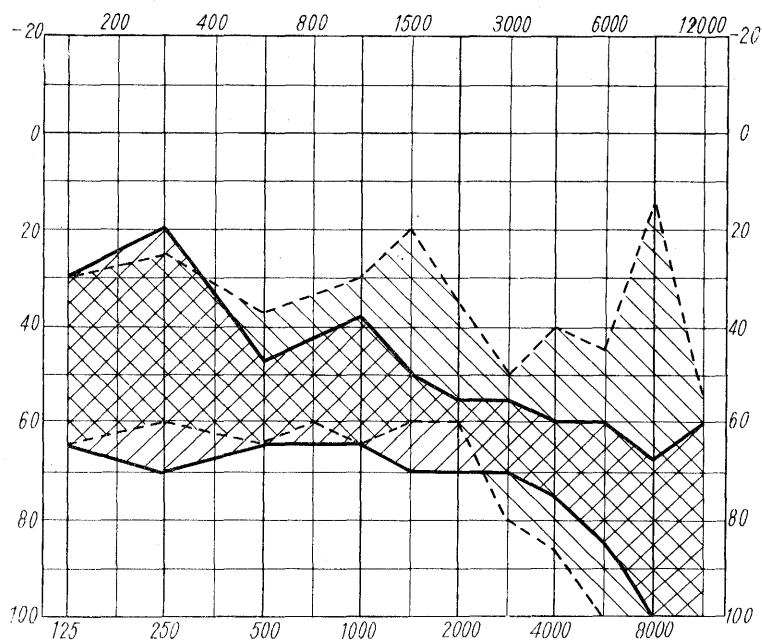


Fig. 3

plication of words and consequently a clinical feature of amnesic aphasia after several months' recuperation. At the same time, it is noted that his loss of hearing for high tones, which has been regarded as irreversible, shows an alarming improvement as illustrated in Figure 4, where the full line indicates the first

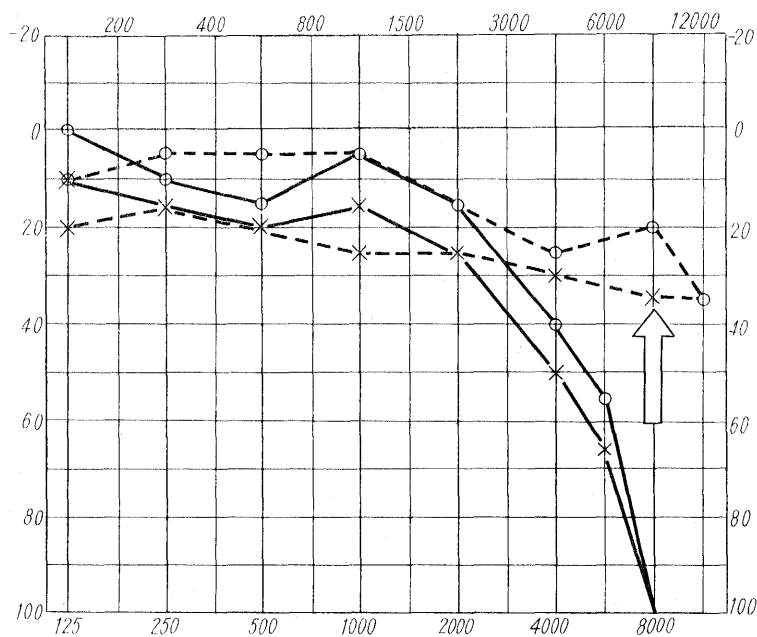


Fig. 4

result and the broken line the latest result. Another case of sensory aphasia has finally presented a clinical state resembling rather subcortical than cortical sensory aphasia as a result of disappearance of paraphasia and verbal amnesia. Nevertheless, hearing loss for high frequencies has remained completely unchanged in contrast with the former case.

A marked improvement of threshold rise for wide frequency range has been found in parallel with that of clinical symptoms in two cases of cortical motor aphasia, of whom one shows a clinical feature of anarthria and one a residual symptom like amnesic aphasia after a considerable time interval.

DISCUSSION AND CONCLUSION

In case of sensory aphasia, the author has obtained the results completely agreeable with those of Alajouanine et al. The patients with sensory aphasia have presented a selective auditory threshold rise for high frequency above 2,000 c.p.s. without exception. It is needless to say that there is no significant abnormality on examination of the ears, nose and throat. Therefore, there is nothing for it but to attribute the threshold rise whether reversible or not to the cerebral damages responsible for sensory aphasia.

As to motor aphasia, the author distinguished three varieties while Alajouanine et al. divided into four groups as follows:

- 1) Hearing for low and middle tones is intact in spite of a severe high tone loss. The audiogram shows a steep slope similar to that of sensory aphasia.
- 2) Loss of hearing is notable for the whole of the tested frequency range. For low and middle tones, threshold rise varies from 20 to 30 db., and for high tone above 2000 c.p.s., the slope becomes much steeper.
- 3) Hearing loss is severe and the audiogram resembles that of conduction deafness. The threshold rise is however more predominant for high tone than for low and middle tones.
- 4) Hearing for low tone is lost in region of 10 db., for middle tone normal but for high tone much more severely affected. In short, a marked threshold rise for higher frequencies than 2,000 c.p.s., which is commonly found to all the four groups, is formerly stated as the characteristic of threshold rise in sensory aphasia.

In the present author's patients with motor aphasia, however, there was only one case whose auditory threshold rise was identical with those of patients of Alajouanine et al. The other eleven showed different types of auditory disorders, which are characterized by absence or slightheadness of loss for high tones.

In order to understand the reason why the results in sensory aphasia agree and those in motor aphasia disagree with the results of Alajouanine et al, it is indispensable to make a comparison of the clinical features between the author's patients and the Alajouanine's, because the results obtained with the aid of clinical

audiometer are the undeniable facts. There may be some differences in interpreting motor aphasia between the author and Alajouanine et al.

At the beginning of this paper, it is stated that the old-fashioned terminology is employed in description of aphasia and that confusion is increased by the fact that different investigators have been animated by different motives and prepared different classifications with different terminology. It is conceivable that the definition of motor aphasia, especially of Broca's aphasia, is differently accepted since Marie's contention in France.

In brief descriptions of clinical symptoms of Alajouanine's patients with motor aphasia, presence of hemianopia, ideo-motor apraxia and disturbance of comprehension, which suggest presence of a diffuser and greater damage of the cerebral cortex than those in the author's patients, is frequently found. From this fact, it seems that Alajouanine et al. have applied the term Broca's aphasia to more global disorders of speech than the original meaning, which indicates cortical motor aphasia in classification of Wernicke and Lichtheim. Most of the patients with motor aphasia in the study group of Alajouanine et al. may be classified as mixed type of motor and sensory aphasia in our clinics for this reason. If so, it is a matter of course that there is a property common to both the two types of aphasia as they have pointed out.

According to Penfield, electrical stimulation to the first temporal convolution, which has been believed as the most responsible area for sensory aphasia, produces auditory sensations as well as that to Heschl's transverse gyrus. As these two convolutions situate closely adjacent to each other, it is scarcely possible that only Wernicke's area is damaged and Heschl's gyrus remains intact in practical cases of head injury or vascular accident. This is the reason why the patients with sensory aphasia present the auditory disturbances without exception.

In case of motor aphasia, hearing loss can also be explained in a similar manner to that in sensory aphasia. There is, however, a little greater distance in cerebral topography between Heschl's gyrus and Broca's area than between Heschl's and Wernicke's areas, so that the results of assessment in motor aphasia are probably not so identical as in sensory aphasia.

The cochlea is projected in a topographically localized manner to different portions of the medial geniculate body, therefore to be distributed in orderly sequence upon the primary auditory cortex situated in the transverse gyrus, if the results of Woolsey and Walzl in the cat and of Tunturi in the dog are confirmed in man. High tones, to which the base of the cochlea is most responsive, is therefore projected to a different part of the auditory cortex than is low tones from the apex of the cochlea. It is also conceivable that the high tone loss in sensory aphasia and low tone loss in motor aphasia correspond with the topographically distributed projections to auditory cortex, but this conclusion must be reserved until when some information on this organization in man become

available.

Appearance of a cortical deafness, as generally accepted, requires the simultaneous bilateral destruction of auditory cortex because a unilateral disturbance of auditory function can be compensated by the intact cortex in contralateral hemisphere.

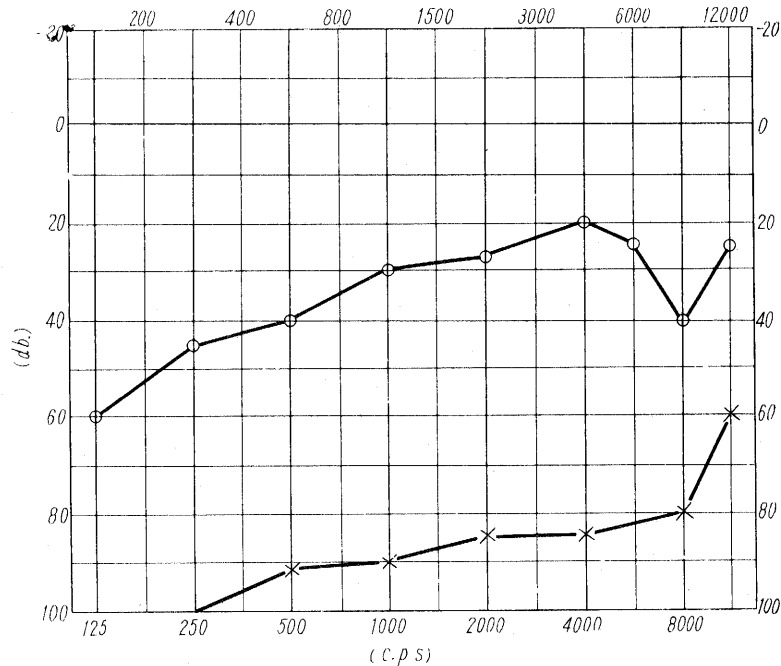


Fig. 5

Figure 5 shows an audiogram recorded in a case of brain abscess located in sylvian fissure of left hemisphere. Hearing loss is found for almost all the tested frequency range even in right ear which has been confirmed to be healthy. According to Penfield again, when auditory cortex is unilaterally stimulated, patient most often refers the sounds to the opposite sided ear and occasionally presents deafness instead of noise. These facts suggest the possibility of unilateral dysfunctioning of auditory cortex to inhibit the function of contralateral hemisphere even if it is just transitory.

It must be noted that the patient showing a marked threshold rise for continuous pure tone has presented less hearing loss or sometimes normal hearing for interrupted pure tones. Most of the otologists maintain that hearing loss does not exist in such a case and that the results obtained with continuous tone testing are not reliable. They may be right. By the way, almost all the patients in the present study have never complained of any auditory disturbance in their daily lives at least but of expressive or comprehensive disorders in speech. If there is an apparent troubles of hearing, the disorder of comprehension can not

be a subject of discussion as a sensory aphasia. Auditory threshold rise for continuous pure tone may indicate a disturbance of auditory attention or an impairment of awareness in patients with aphasia. From the aphasiological view point, this dissociation is important as the property of disorders due to cerebral damages.

The functional oscillation illustrated in Figure 3 also confirms that this kind of hearing loss in aphasia is of central origin. For all that, it is impossible to attribute aphasia to the auditory disturbance or the functional oscillation of hearing. As shown above, the average loss of hearing for speech frequencies is less than 10 db. in case of sensory aphasia, and loss for higher frequencies than 2,000 c.p.s. has no important influence upon the comprehension of spoken words.

The fact that the auditory threshold rise modifies in parallel with the improvement of clinical disorders indicates the topographical distribution of responsible areas to unit disorder in the brain. The author does not intend to determine any strict localization of speech disorders but to clarify the anatomo-clinical correlation of cerebral functions.

Ohashi claims that an accurately circumscribed lesion of Wernicke's area causes a clinical feature of pure word-deafness but not of Wernicke's aphasia. He divides a sensory aphasia into three unit disorders; namely agnosic, amnesic and paraphasic, as some earlier investigators intended. Furthermore, he intends to correlate them with different portions in temporal lobe; agnosic disorder with Wernicke's area, amnesic disorder with posterior portion, and paraphasic disorder with insula and adjacent area.

It is a matter of course that all the brain damaged present in their acute stage disorders not only due to destructed area but also due to adjacent area and sometimes all the regions that receives nerve fibers from destructed area. Some of the remote symptoms may be subclinical and not apparent, but most of the clinical features must be a heterogenous collection of unit symptoms of different pathology according to Bay's expression. A speech disorder cannot be exceptional. Sensory aphasia is a collection of three unit disorders such as above mentioned, while motor aphasia is that of three unit disorders; amnesic, paraphasic and anarthric symptoms. A well balanced collection of these three unit disorders forms a cortical sensory or these three unit disorders forms a cortical sensory or a cortical motor aphasia, particularly its typical case. Therefore sensory aphasia and motor aphasia cannot be essentially different to each other from this point of view. Ohashi and the present author suppose the existence of schema in the background of these unit disorders. About the schema, subconscious mediator between the physiological and psychological events, between sound and meaning in case of speech, between gestalt and symbol in general, the author intends to discuss in another opportunity.

SUMMARY

Twenty-three cases of different types of aphasia have been studied with the aid of pure tone clinical audiometers and revealed the noticeable loss of hearing in sensory and motor aphasia while the patients with amnesic aphasia shows normal hearing. Hearing loss for high frequency in sensory aphasia has been identical but low and middle tone loss in motor aphasia has not been identical with the results in the paper of Alajouanine et al. Explaining the difference of results from the view point of terminology, the author has intended to attribute the threshold rise to an inevitable dysfunctioning of primary auditory cortex in neighbourhood of Wernicke's and Broca's areas. Then, anatomo-clinical correlation and unit symptoms of speech disorders have been discussed.

ACKNOWLEDGEMENT

This paper is dedicated to honour the retirement of Prof. Mitsuzi Goto, one of the leading figures in otorhinolaryngology in Japan. The author wishes to express his gratitude to Dr. Shigekazu Okamoto, Professor of Neuropsychiatry, Kansai Medical School, for his interest and encouragement, to Dr. Hiroshi Ohashi, Lecturer in Neuropsychiatry, University of Kyoto, for his advice and guidance, to the staff of otorhinolaryngological wards of Kyoto University Hospital and of the Kansai Medical School Hospital for making facilities available to carry out the assays.

This paper delivered at the 58th. annual meeting of Japanese Association of Neuropsychiatry held in Okayama, in 1961.

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